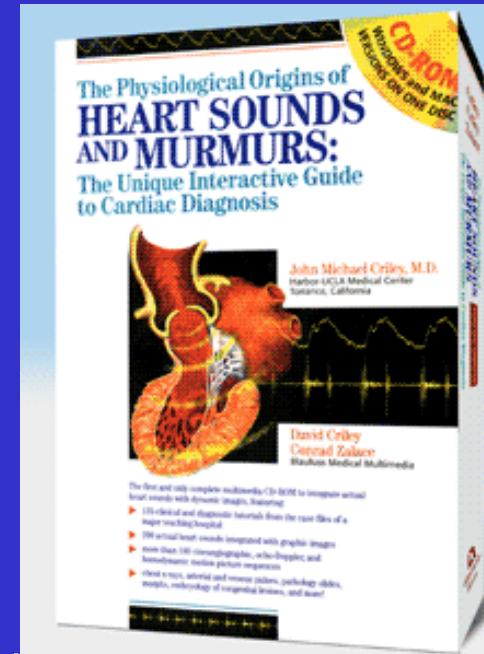


# Introduction to Cardiac Auscultation

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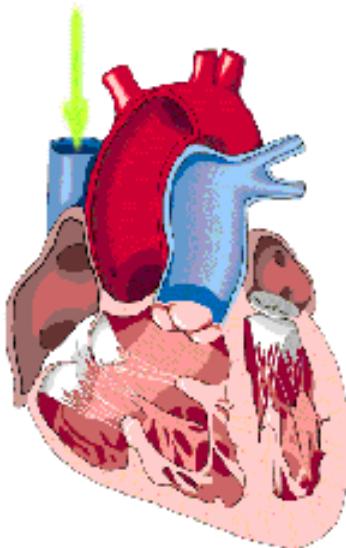
# Overview of Module



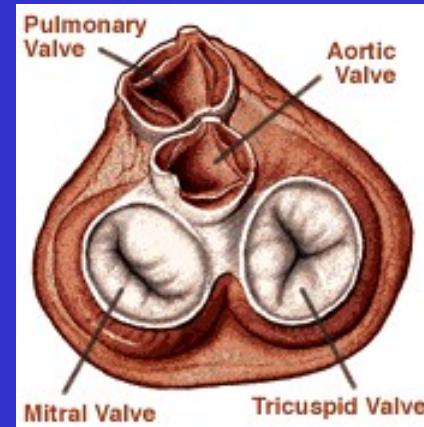
- Today's lecture
  - review of valve disease
    - Aortic stenosis
    - Aortic regurgitation
    - Mitral stenosis
    - Mitral regurgitation
- ICR
  - discussion of cases/physical exam findings 11/30/06
- Lecture
  - 9 hours of auscultation
  - review sessions as desired by class
- Rest of your life
  - practice, practice, practice
  - *NEW!!!* Criley CD now available through internet  
[www.blaufuss.net/USUHS/tutorial/](http://www.blaufuss.net/USUHS/tutorial/)

# Normal Valve Function

- Prevent backward flow of blood
- Permit forward flow of blood
- Valve disease interferes with these functions



- Right Atrium
- Tricuspid Valve
- Right Ventricle
- Pulmonic Valve
- Pulmonary Arteries
- Pulmonic Veins
- Left Atrium
- Mitral Valve
- Left Ventricle
- Aortic Valve
- Aorta



# Abnormal valve function

- Allows backward flow
  - valve is “leaky;” “regurgitant;” “incompetent”
- **Reduces cardiac output while increasing workload**
  - results in inefficient pumping; greater volume of blood needs to be pumped with each beat to maintain cardiac output
  - “volume load”
  - Typically causes dilatation of the cardiac chamber
  - Backwards jet causes turbulence that is audible as murmur

# Abnormal valve function

- Prevents forward flow
  - valve does not open well
  - Greek stenosis, a narrowing
- **Reduces cardiac output while increasing workload**
  - Heart must develop more pressure to move blood
  - “pressure load”
  - usually results in hypertrophy of proximal (“upstream”) chamber (LA in MS, LV in AS)
  - acceleration of blood through tight valve causes turbulence that is audible as a murmur

# Valvular Disease

## General Principles

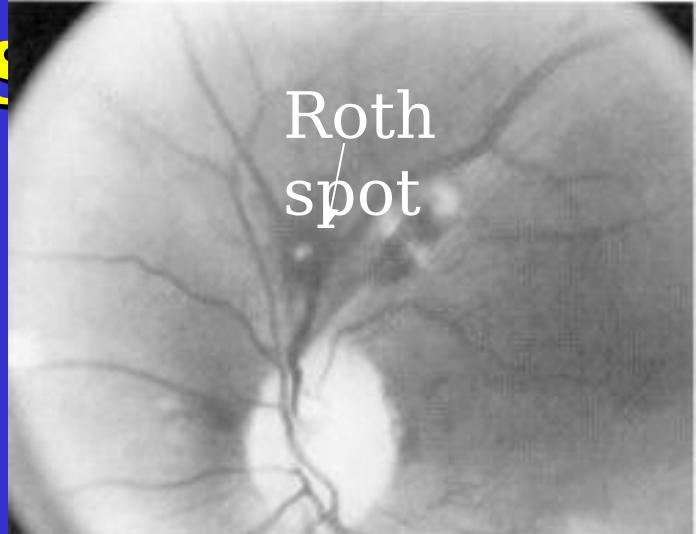
- Left sided valvular disease more prone to cause serious hemodynamic problems
- stenotic lesions cause pressure overload on proximal chamber- concentric hypertrophy (thickened walls)
- regurgitation causes volume overload- eccentric hypertrophy (dilatation)
- stenotic lesions cause symptoms **sooner** than regurgitant lesions but respond to therapy better

# Valvular Disease

- Rheumatic fever
  - regurgitation frequently present *acutely*
  - long term predominant effect is stenosis
- Endocarditis causes regurgitation
  - patients with valve dz should take antibiotics prior to dental work to prevent endocarditis
- All patients with symptomatic valvular disease (i.e. dyspnea, chest pain, syncope) need to be evaluated for surgical correction
  - Some asymptomatic subjects also need correction “before it’s too late”

# Endocarditis

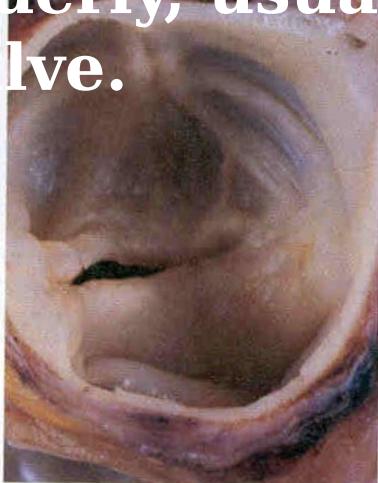
- **Etiology**
  - damaged valve (RHD) exposed to bacteria in blood stream
  - *S. viridans, S. aureus*
- **Clinical**
  - acute, subacute, chronic
  - fever, murmur, ESR
  - (+) blood cultures
- **Treatment**
  - antibiotic according to organism
  - future prophylaxis for procedures



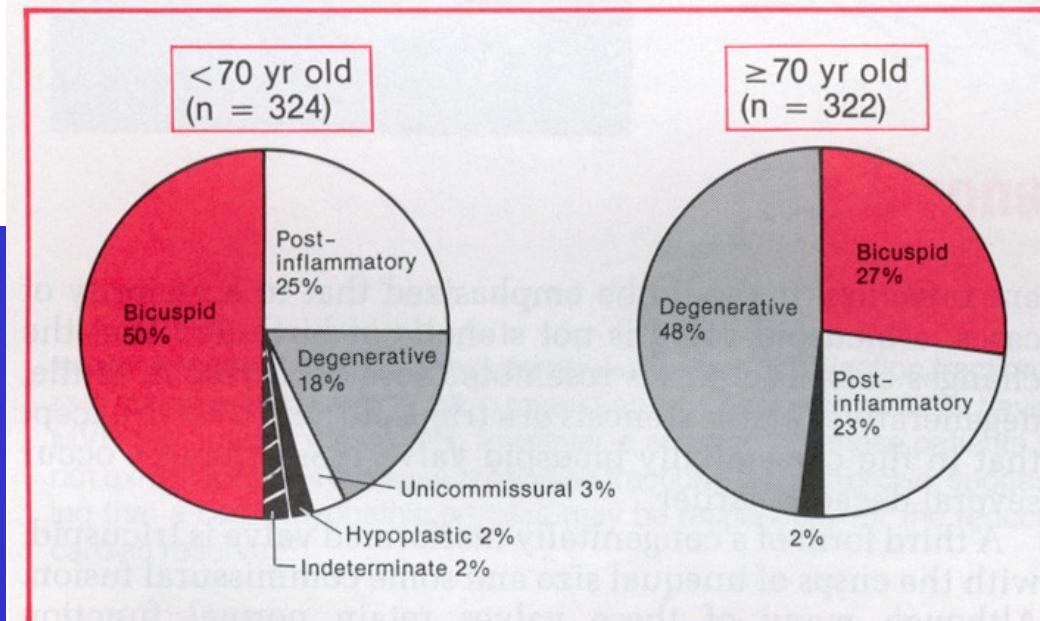
# Valvular Aortic Stenosis

- failure of valve to open normally during systole, requiring LV to develop excess pressure to overcome increased resistance
- pressure gradient between LV and aorta may be as much as 100 mm Hg
- causes concentric hypertrophy
- symptoms of exertional chest pain, syncope, dyspnea
  - mandate valve replacement to prevent sudden death

**Aortic stenosis due to bicuspid valve.**  
**Symptomatic AS in young usually due to congenitally abnormal valve or (less frequently in US) rheumatic disease. In elderly, usually due to calcification of the valve.**

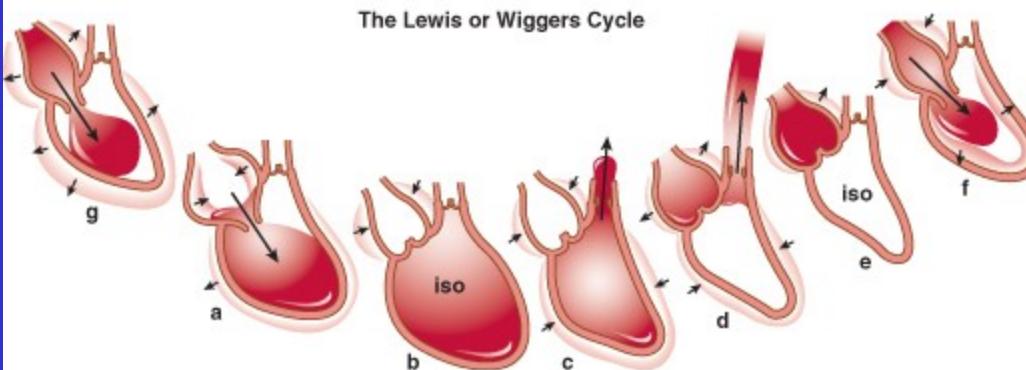
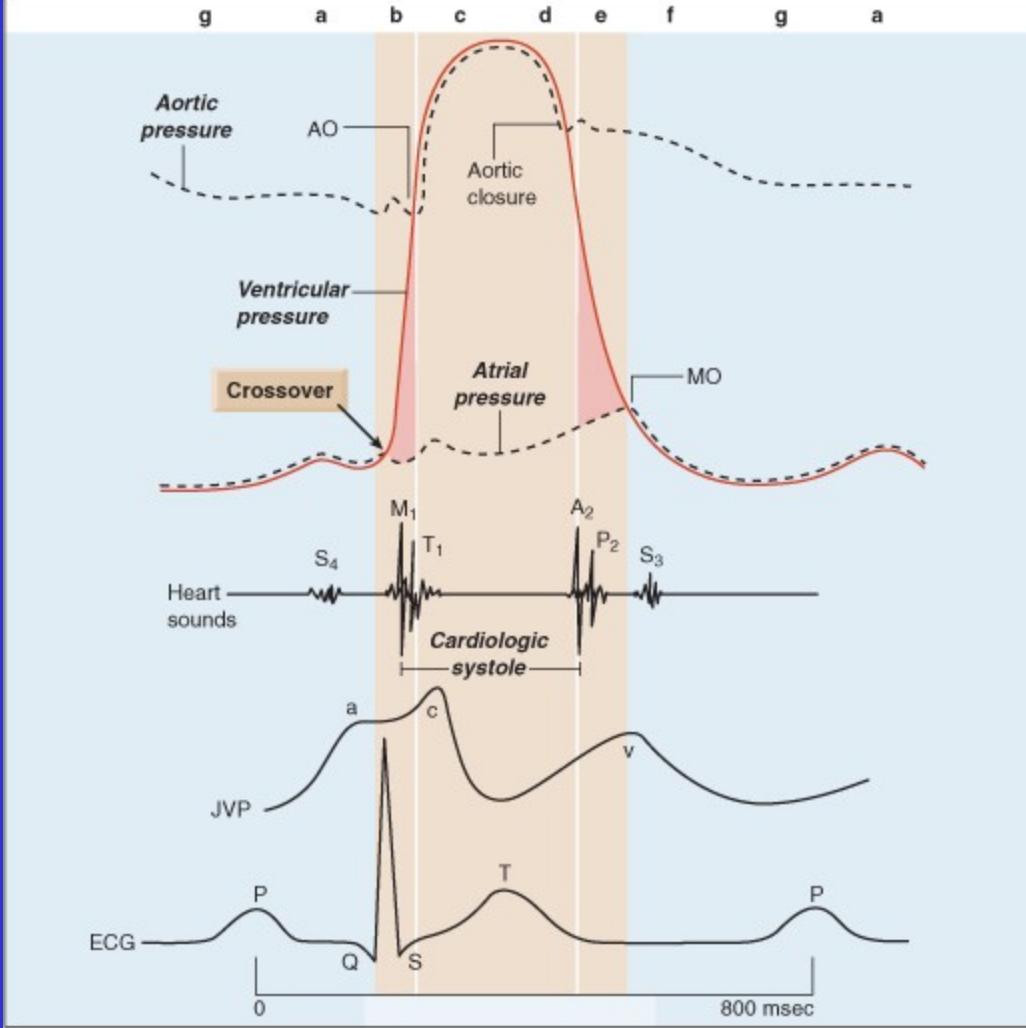


**Fig. 29-24 Aortic stenosis.** Mild stenosis in valve leaflets of a young adult. (From Damjanov I, Linder J, editors: *Anderson's pathology*, ed 10, vol 1, St Louis, 1996, Mosby.)



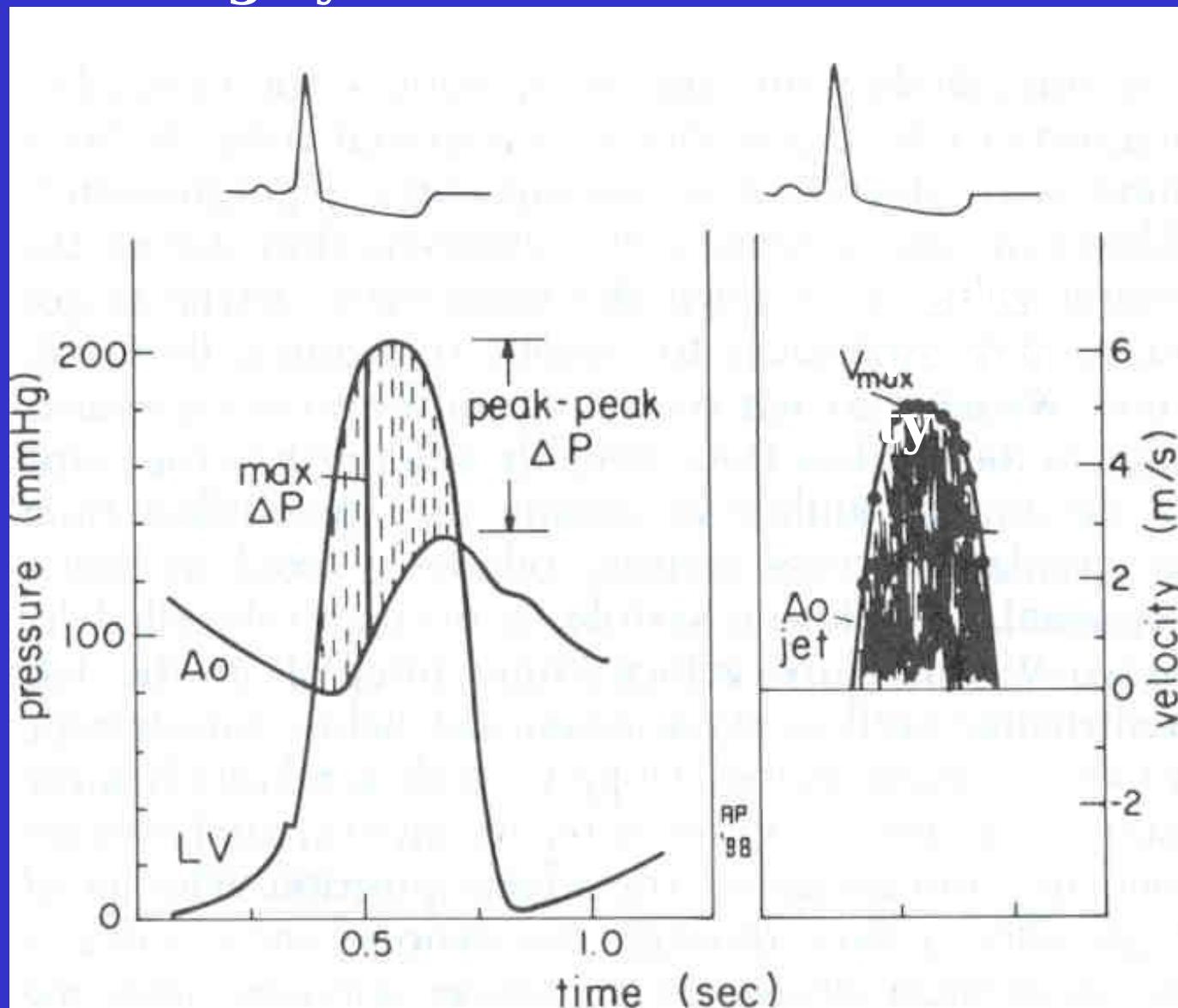
# Grades of AS

- Normal valve area 3-4 cm<sup>2</sup>
- Mild AS >1.5 cm<sup>2</sup>
- Moderate >1.0 cm<sup>2</sup>
- Severe AS when area  $\frac{1}{4}$  normal
  - <1 cm<sup>2</sup> for large person
  - <0.75 cm<sup>2</sup> for normal person

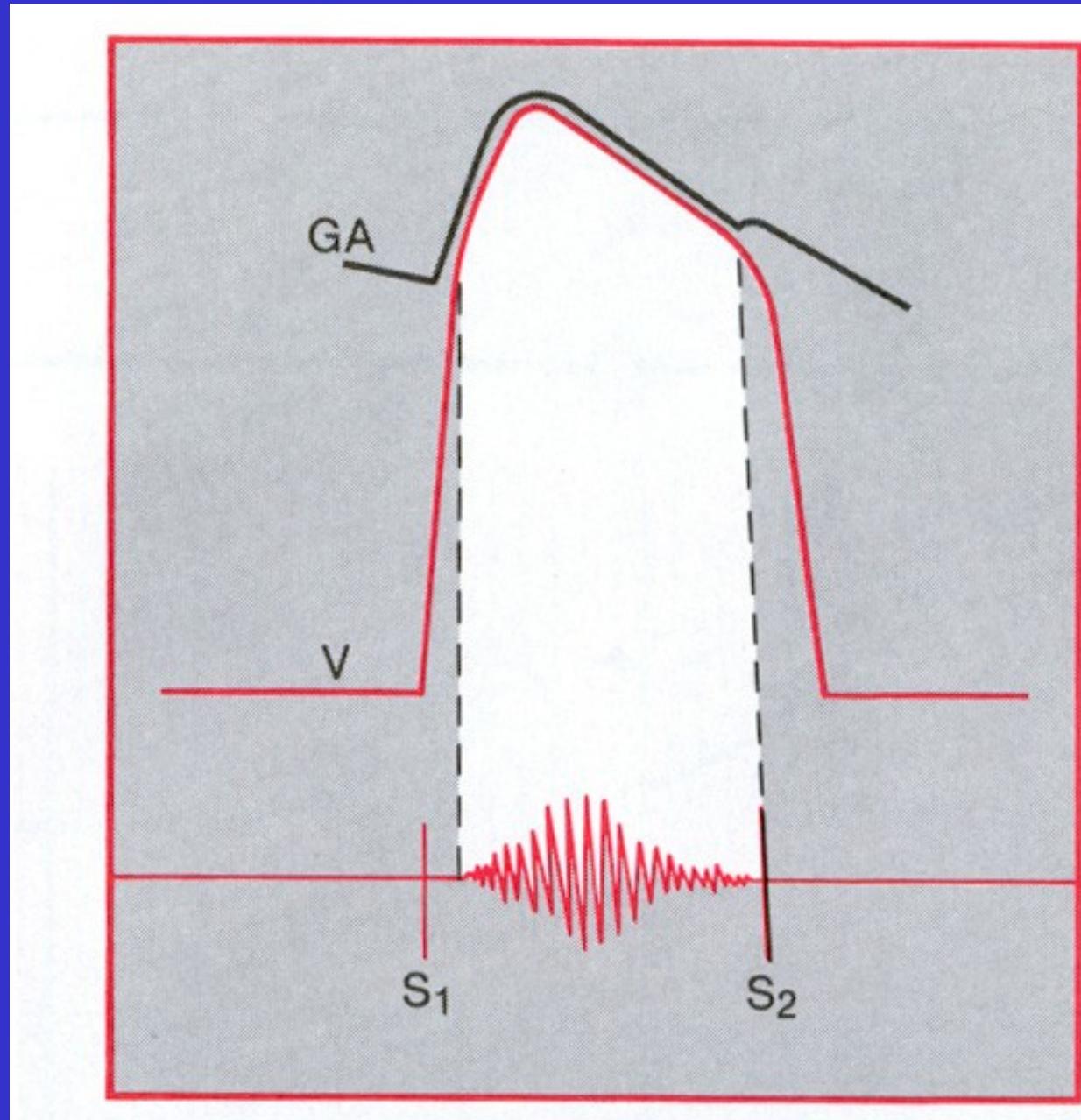


# Pressure gradient develops between LV and Aorta during systole in AS

Note delayed upstroke of aortic pressure; murmur peaks with max pressure gradient due- equals time of greatest blood velocity

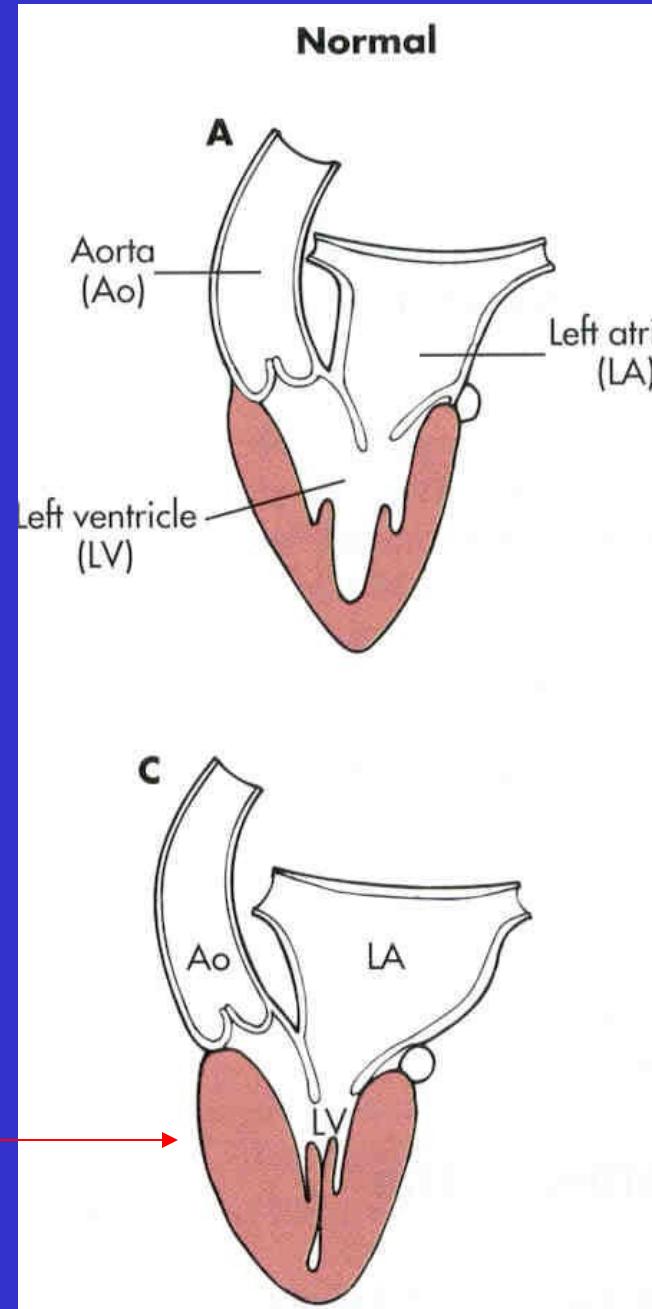


**Murmur in AS is mid-systolic, crescendo-decrescend**  
o. Note that it begins AFTER S1 and ends BEFORE S2

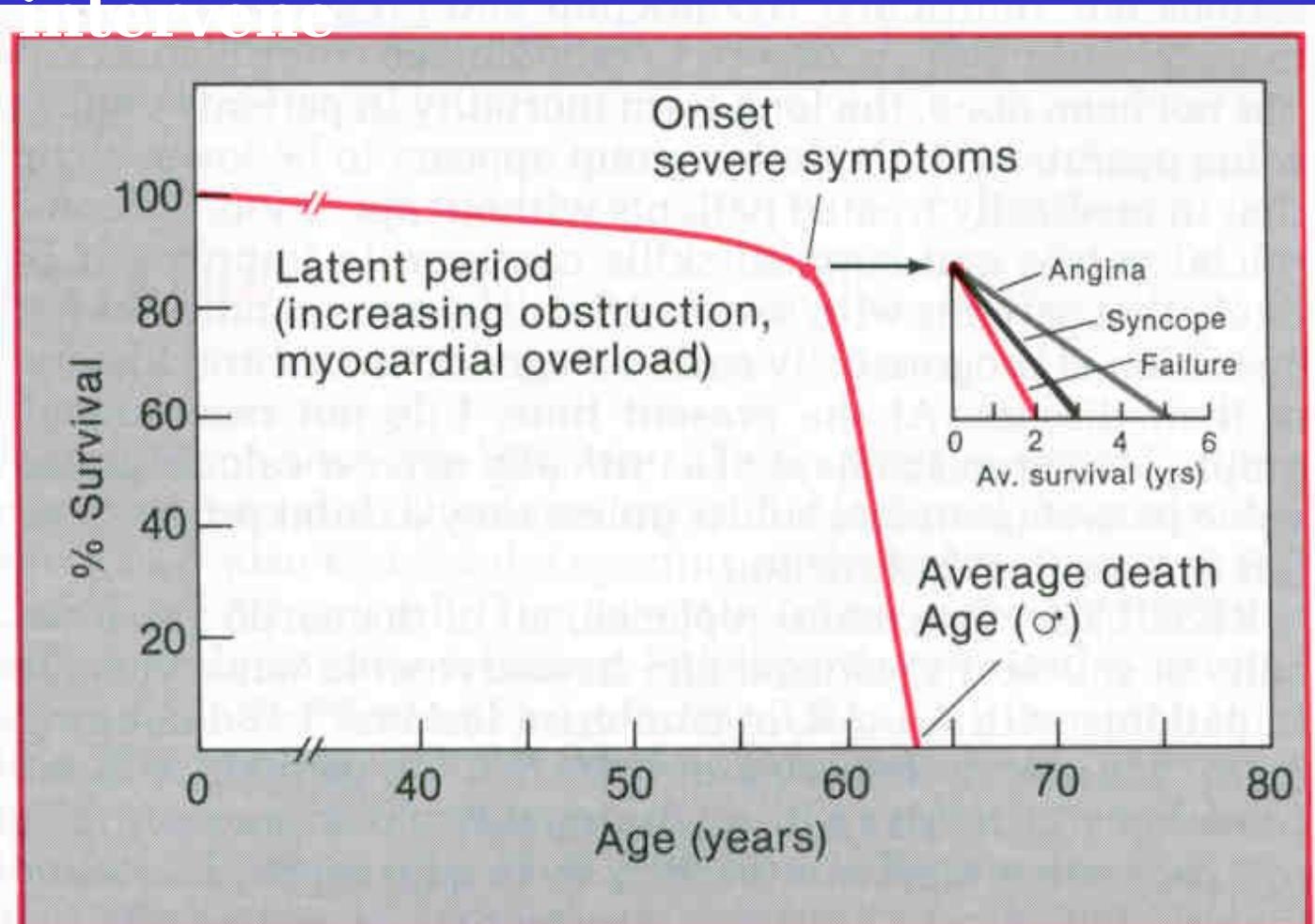


Comparison of cross section of normal left heart with heart showing concentric left ventricular hypertrophy. Note reduced chamber size, thickened walls, enlarged left atrium.

Causes include hypertension, aortic stenosis (pressure overload), inherited myosin disorder (HOCM)**Concentric hypertrophy**



## Rapid fall in survival once symptoms intervene



**FIGURE 34-27.** Natural history of aortic stenosis without operative treatment. (From Ross, J., Jr., and Braunwald, E.: Aortic stenosis. *Circulation* 38[Suppl. V]:61, 1968, by permission of the American Heart Association, Inc.)

# Symptomatic AS- management

- NO SAFE MEDICAL RX for Severe AS
- Physical diagnosis straight forward
- systolic crescendo-decrescendo murmur
  - loudest in aortic area usually (sometimes apex)
  - radiates to carotids
- LV hypertrophy associated with gallop (S4)
- Signs of critical AS
  - carotid upstrokes small and delayed in severe AS
  - loss of aortic component of S2
  - late peaking murmur

# Mitral Stenosis

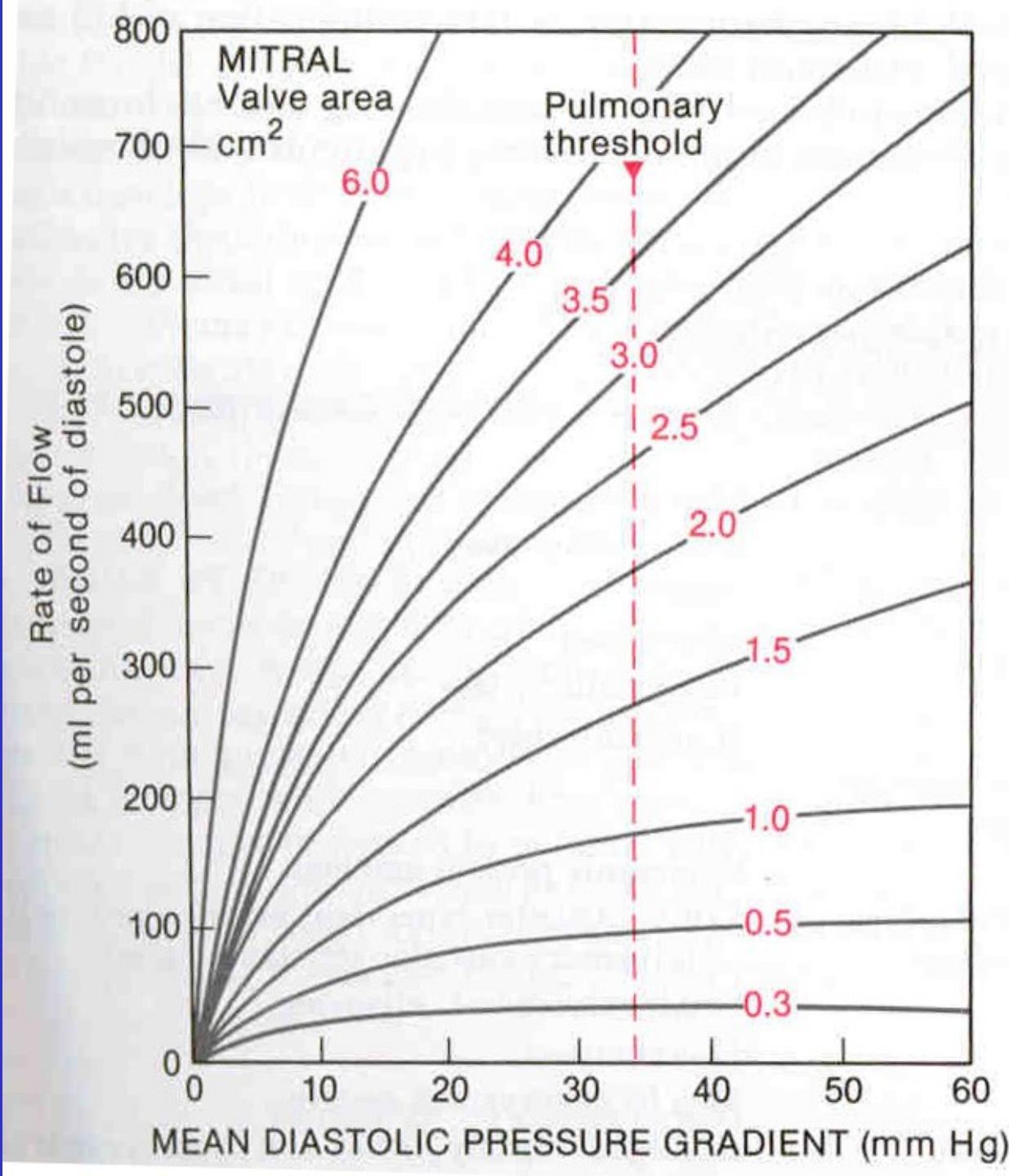




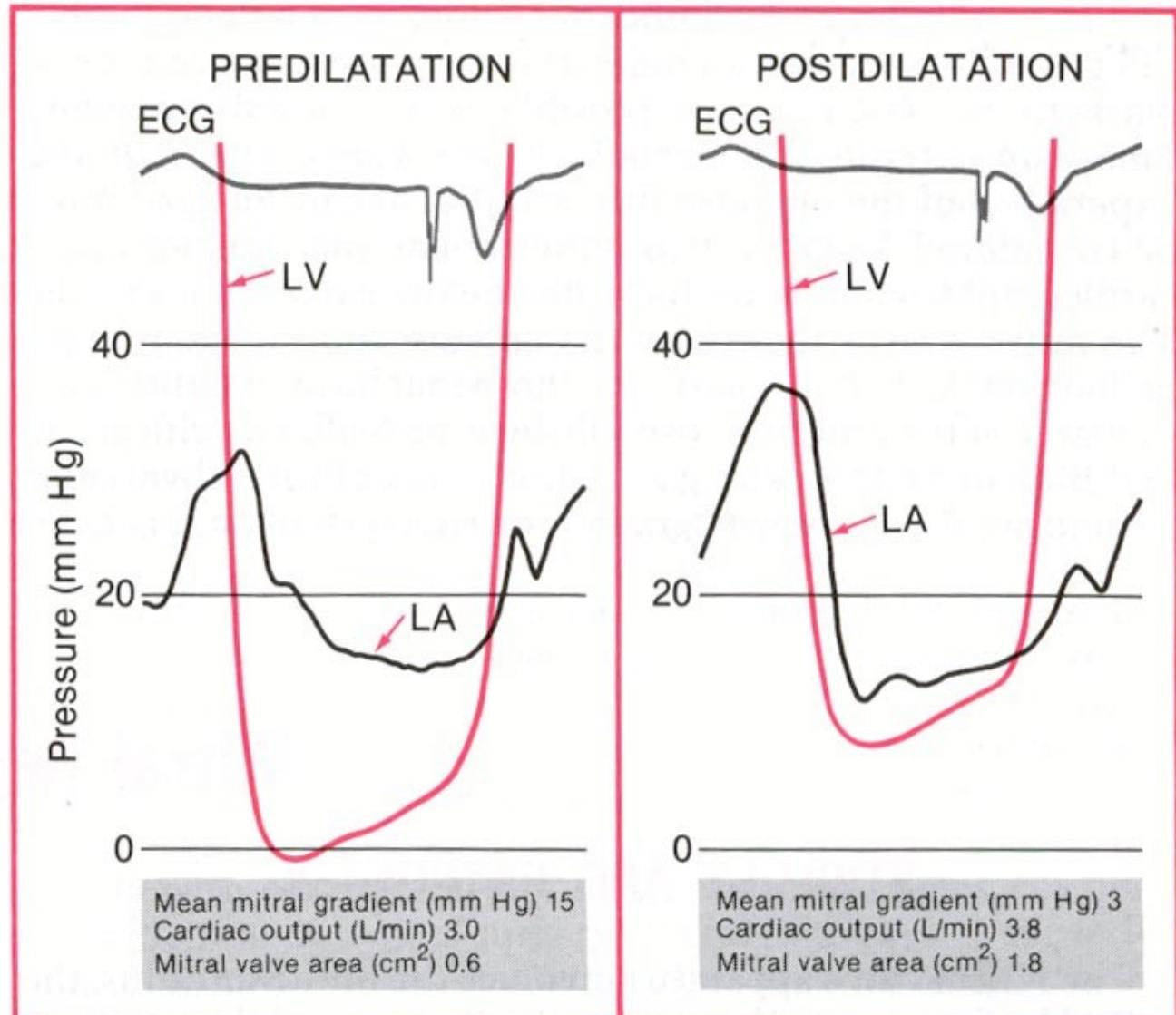
# Mitral Stenosis

- Almost always rheumatic in origin
- Murmur may be subtle, but high flow states cause increased pressure gradient, pulmonary edema
  - classic presentation is during vaginal delivery. Tachycardia, straining, volume increase cause pulmonary edema
- Patients eventually have exertional dyspnea, atrial fibrillation (often with thromboembolism), chest pain

**Normal MVA 4-5 cm<sup>2</sup>**  
**>2.5 cm<sup>2</sup> asymptomatic**  
**<1.5 cm<sup>2</sup> may have sxs at rest**  
**Stress which increases transmitral flow or decreases diastolic filling time will significantly increase gradient**



**MS causes high pressure in LA, low pressure in LV, so poor LV diastolic filling and backward failure symptoms of pulmonary edema, atrial fibrillation, and thromboembolism**  
**Surgery here**



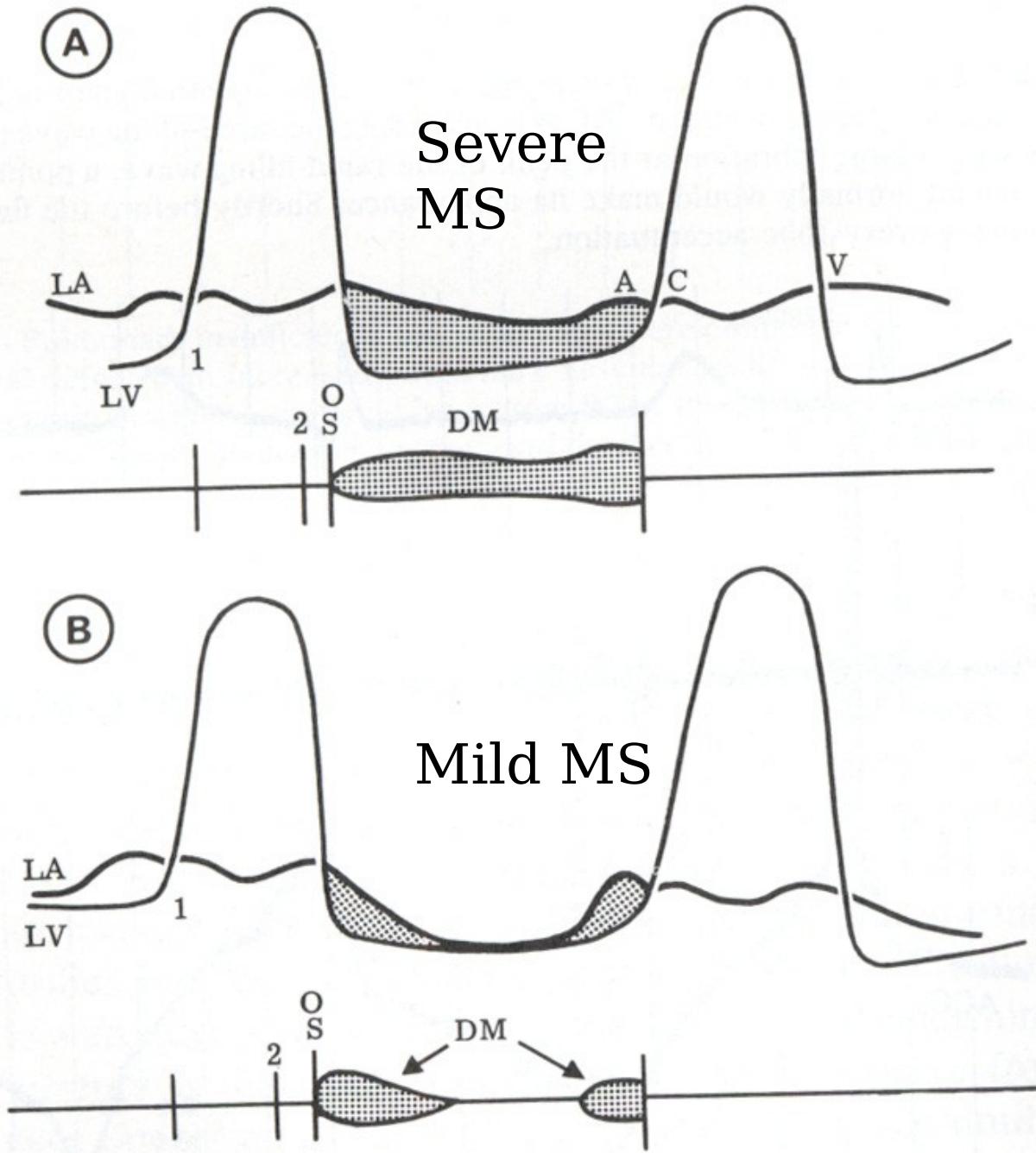
**FIGURE 34-8.** Simultaneous left atrial (LA) and left ventricular (LV) pressure before and after balloon valvuloplasty of the mitral valve in a patient with severe mitral stenosis. (Courtesy of Raymond G. McKay, M.D.)

# Mitral Stenosis

- Turbulent, high velocity flow occurs during diastole
  - murmur is therefore a DIASTOLIC, low frequency rumble heard at apex with stethoscope bell, patient in L lateral decubitus
  - requires quiet concentration, palpate carotid to time systole/diastole
- Always look for MS in patient with new Atrial fibrillation
  - rate control, anticoagulation crucial

## MS Murmur

**Severe MS associated with pan-diastolic rumble, short S2-OS interval.**  
**Mild MS (B) associated with decrescend o-crescendo rumble, longer S2-OS interval**

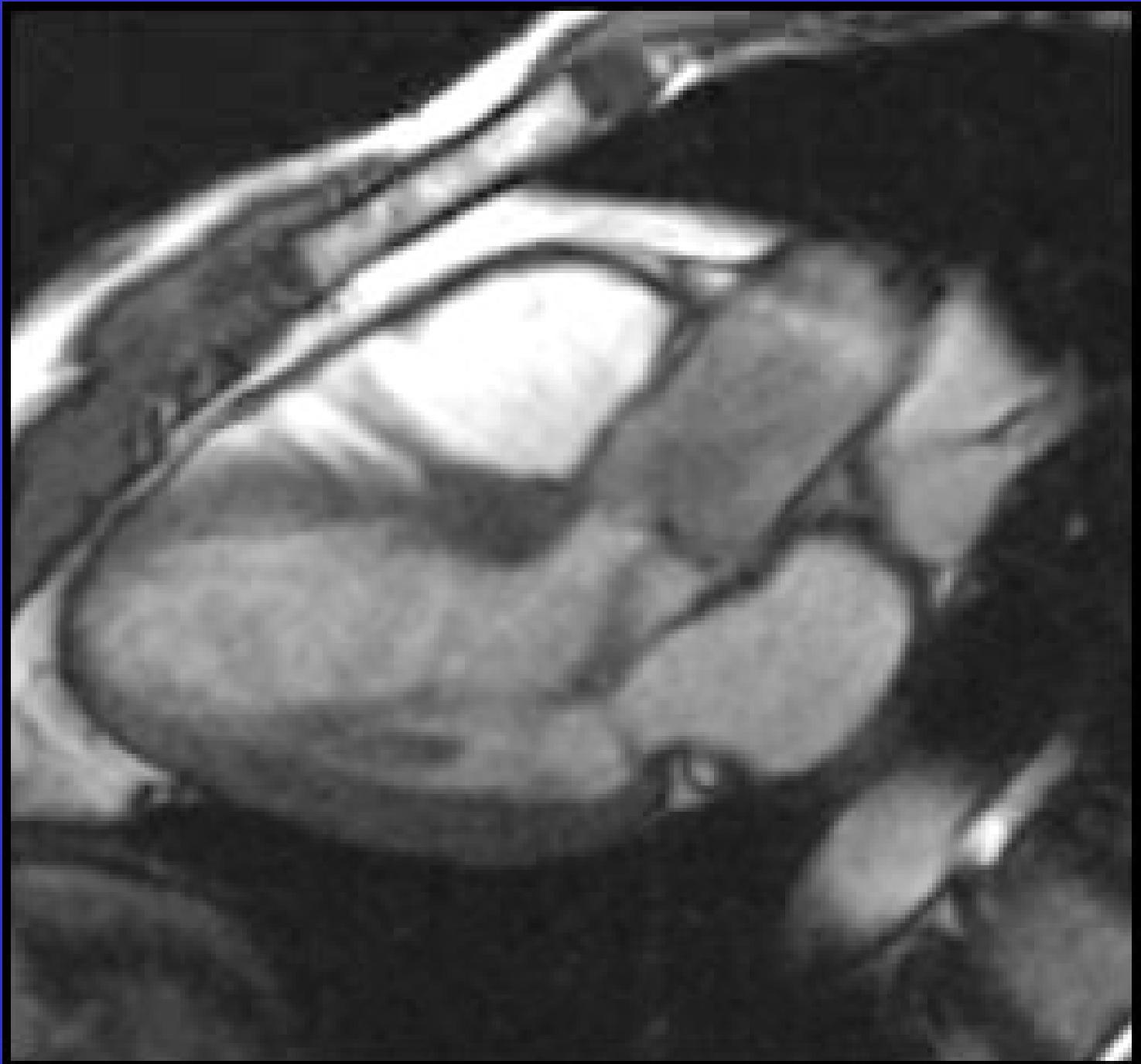


# MS Mortality

- Minimal sxs >80% 10 year survival
- Limiting sxs, <15% 10 year survival
- Untreated patients
  - 60-70% progressive pulmonary edema
  - 20-30% systemic embolism
  - 10% pulmonary embolism
  - 1-5% endocarditis/infection

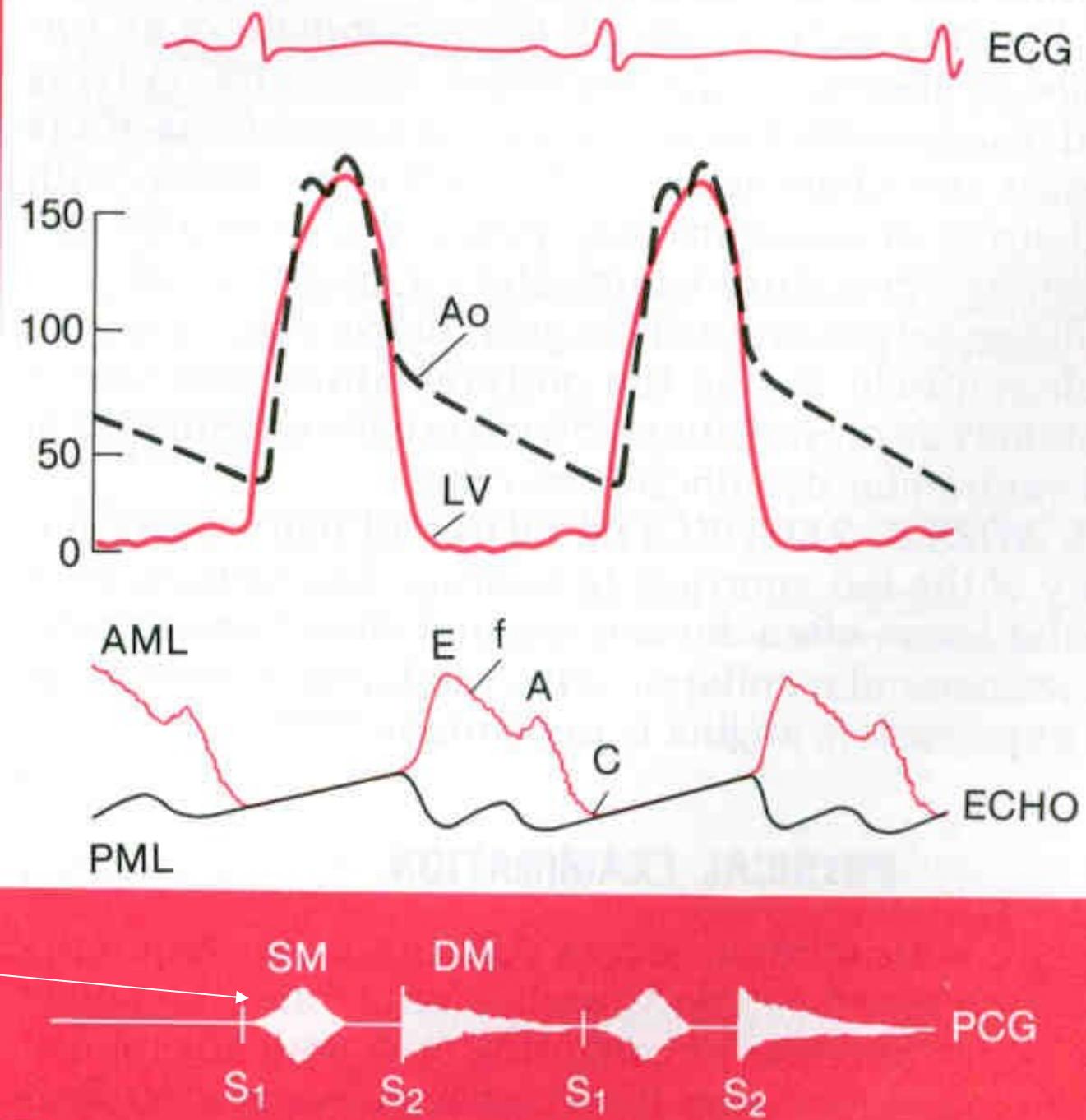
# Aortic Regurgitation

- Loss of cardiac output backwards from aorta into LV
- congenital, endocarditis, age, aortic disease, collagen vascular, syphillis
- Early diastolic, decrescendo murmur best heard at LLSB with diaphragm
  - subtle, have pt lean forward, breathe out
  - associated with wide pulse pressure

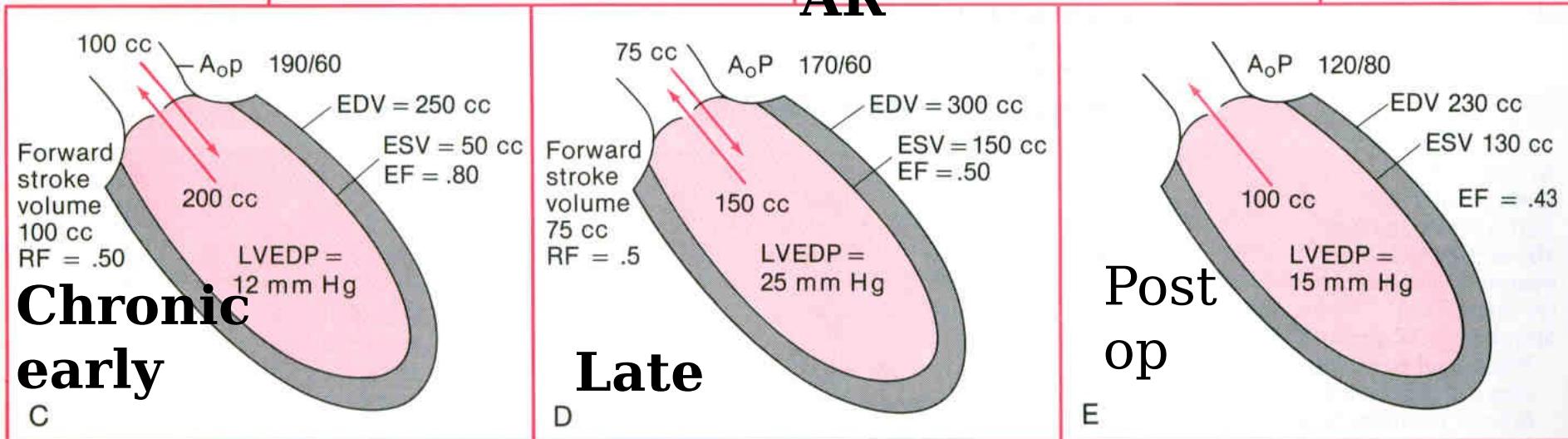
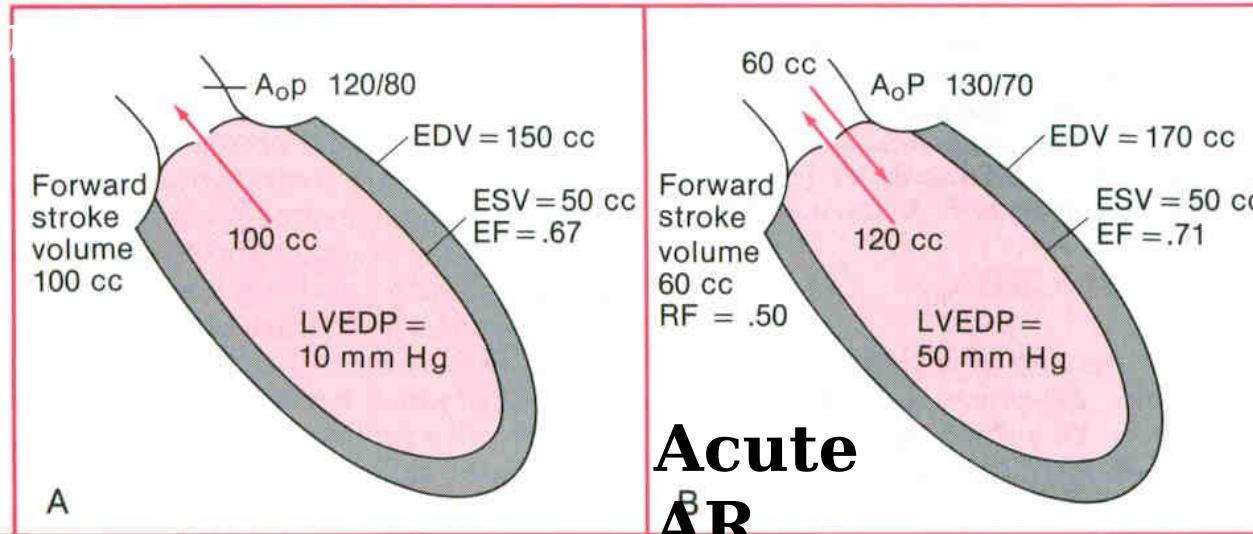


## Chronic AR

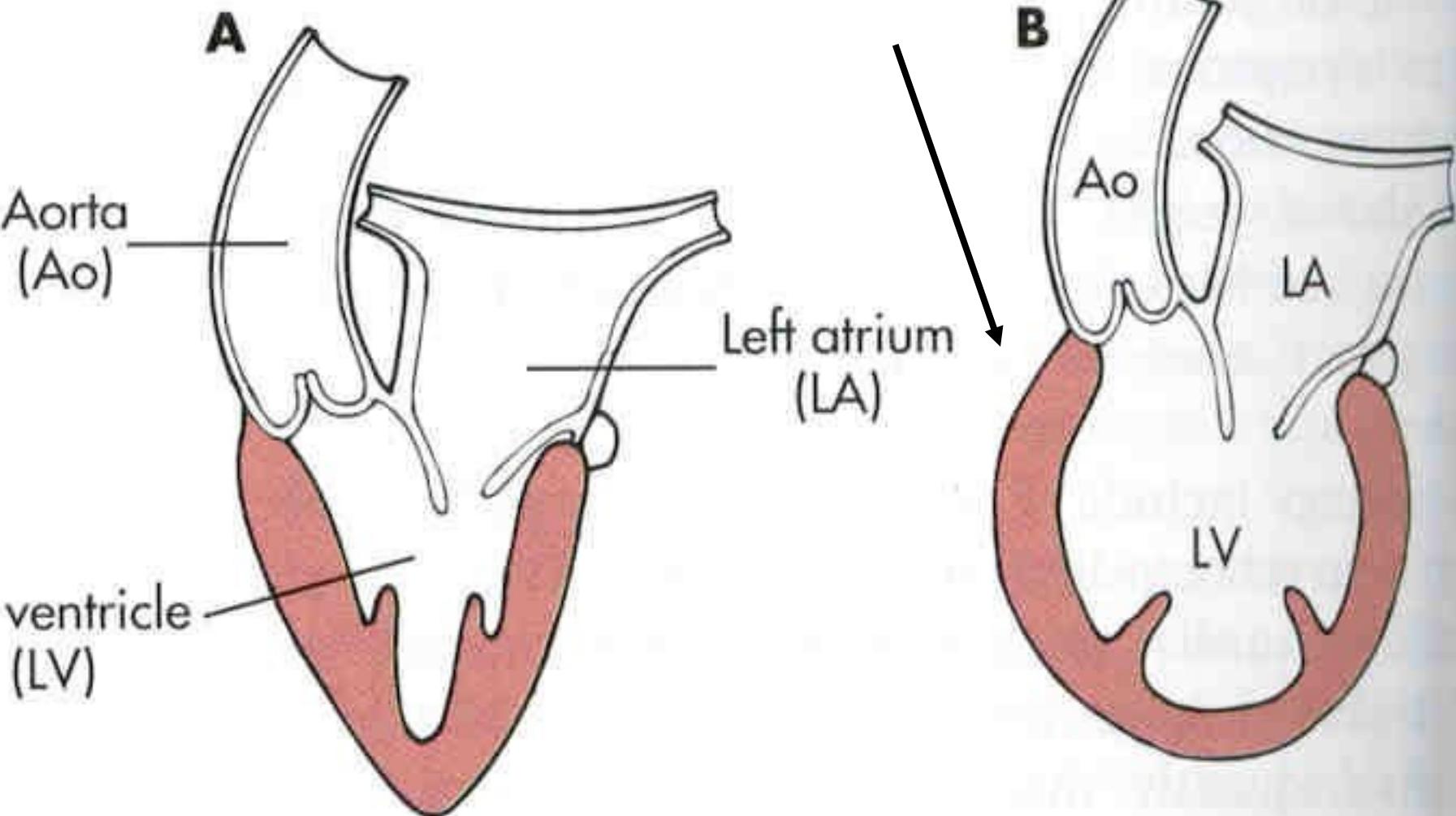
Early diastolic decrescendo murmur at time of greatest pressure difference between Ao and LV. Note early systolic



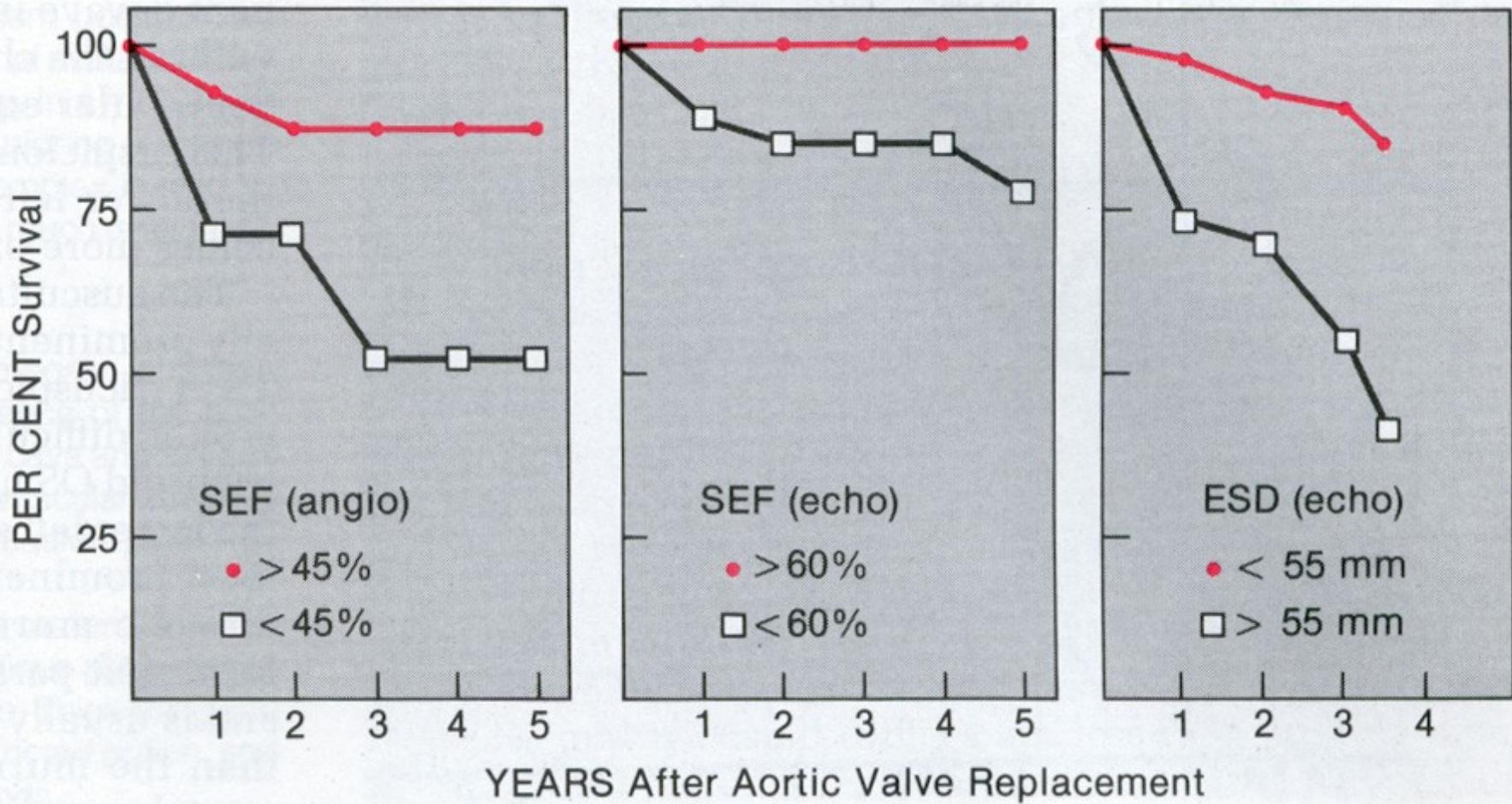
**Acute AR causes sudden increase in LVEDP, pulmonary edema. Over time, eccentric hypertrophy allows LV to accommodate increased volume, but ventricle fails**



# Effect of volume overload



# Survival post AV replacement for Aortic Regurgitation based on Pre-op Systolic function



# Mitral Regurgitation

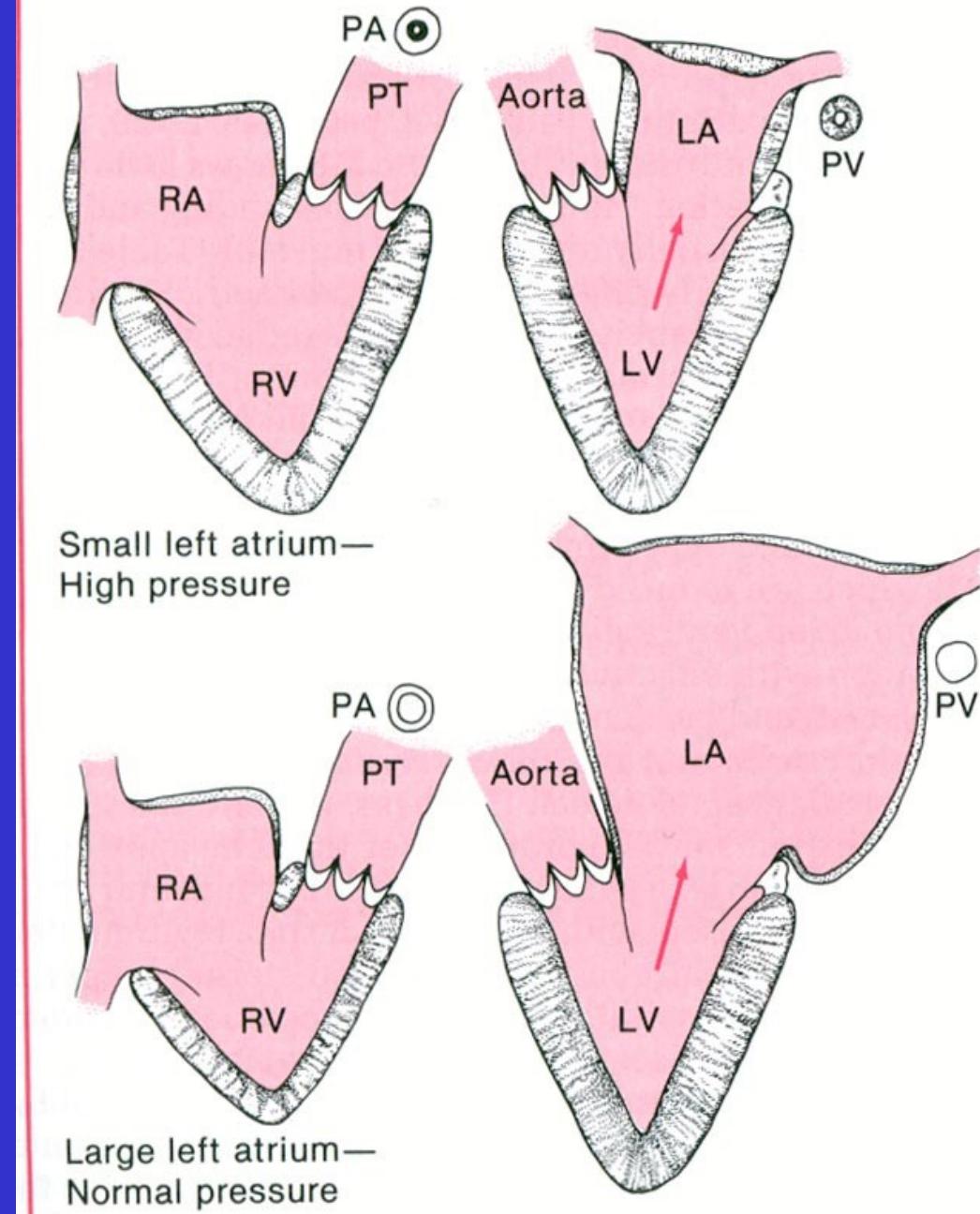
- Incompetent mitral valve allows loss of stroke volume back into LA
- Mitral valve prolapse most common cause
  - rheumatic disease and endocarditis
- PE much less subtle than MS
  - loud pan-SYSTOLIC murmur, loudest at apex and radiating into axilla
  - typically soft S1
  - S2 obscured by murmur
  - presence of S3 suggests severe MR



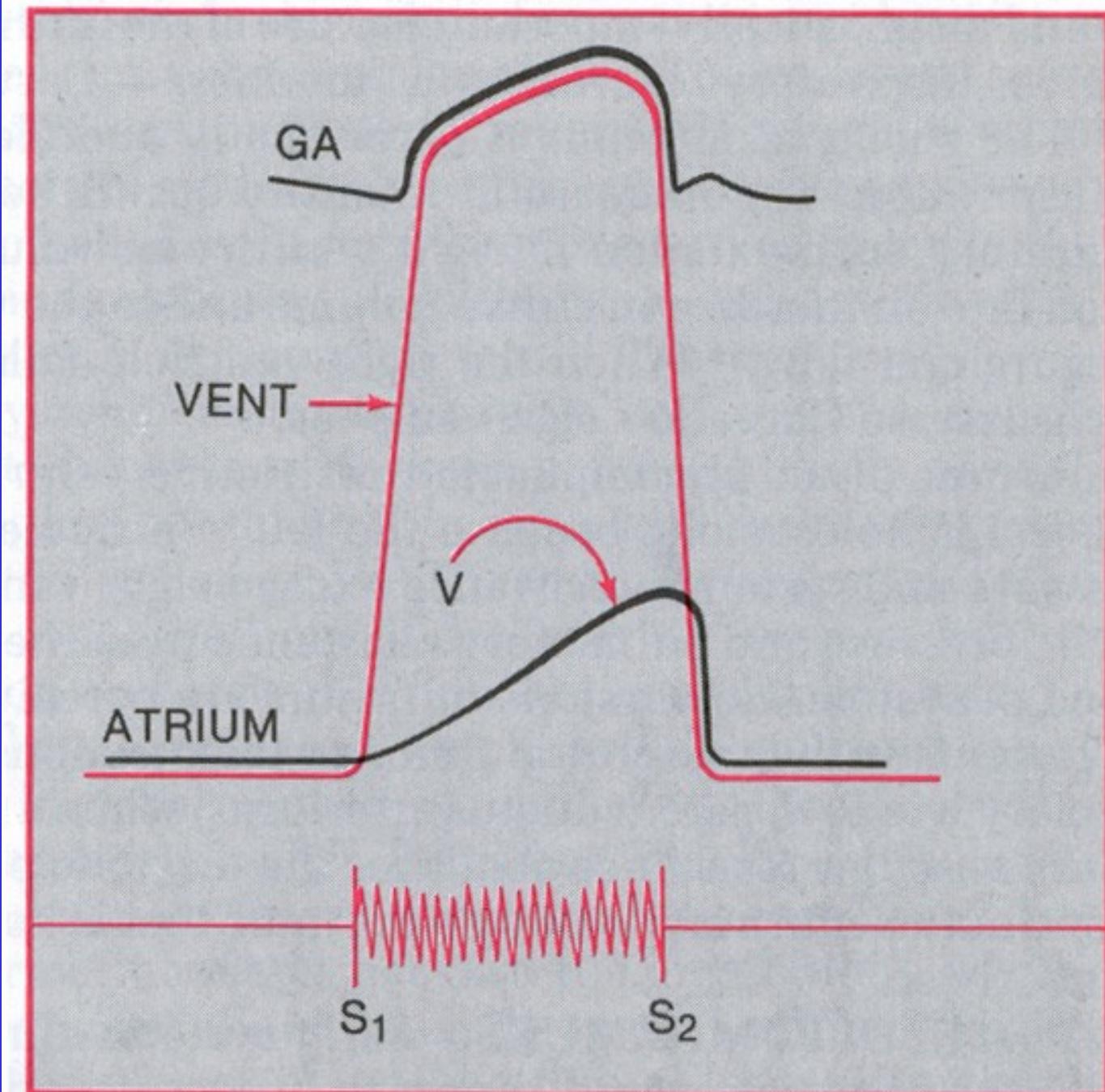
**Acute MR causes immediate pulmonary edema, often fatal post infarction, acute endocarditis, chord rupture**

**Chronic MR causes LA enlargement, eventual LV dilatation and failure.**

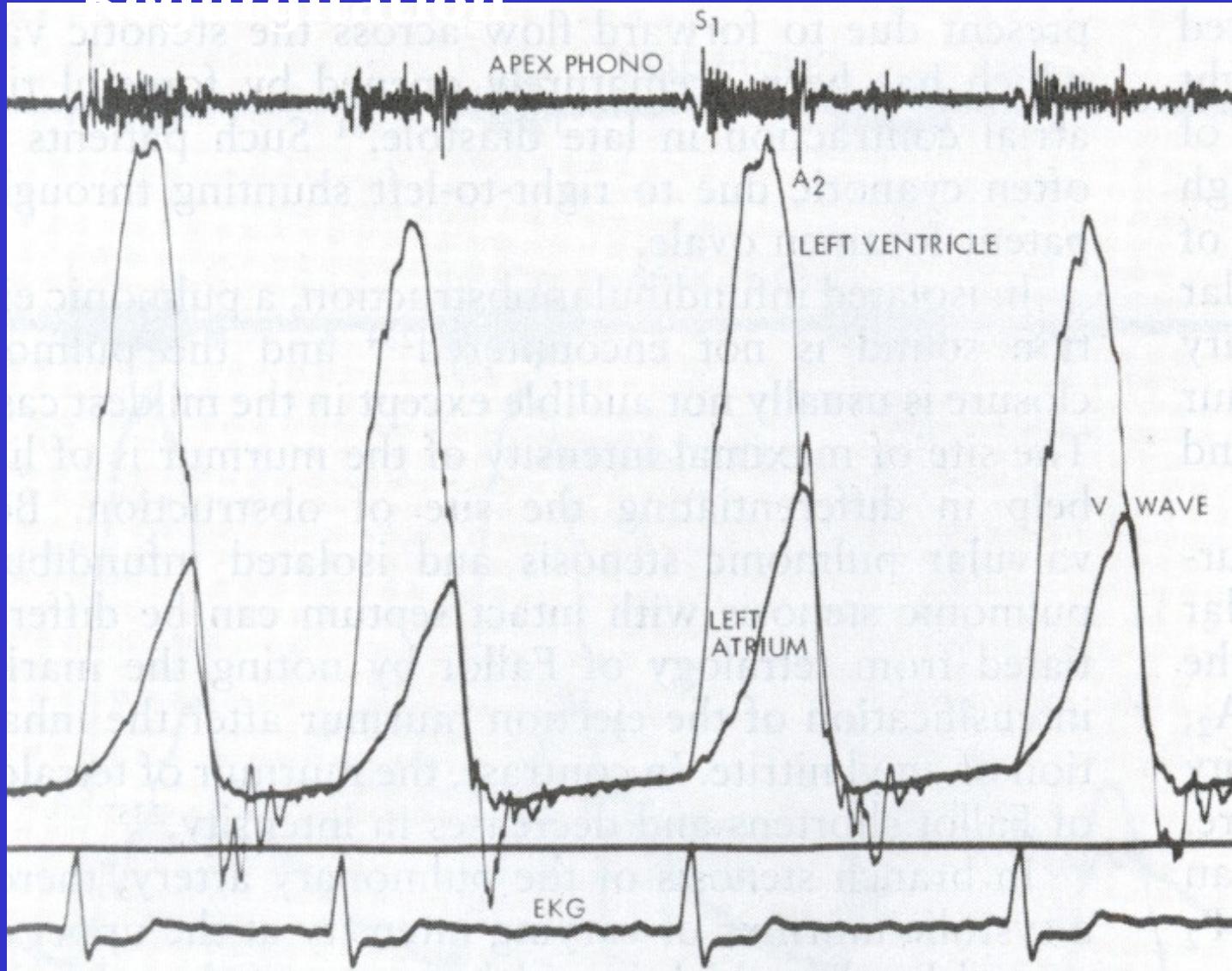
## THE SYNDROME OF MITRAL REGURGITATION



**Murmur in  
MR Note  
Holosystolic  
nature-  
begins with  
S1 and ends  
at S2 (often  
goes  
beyond S2).**



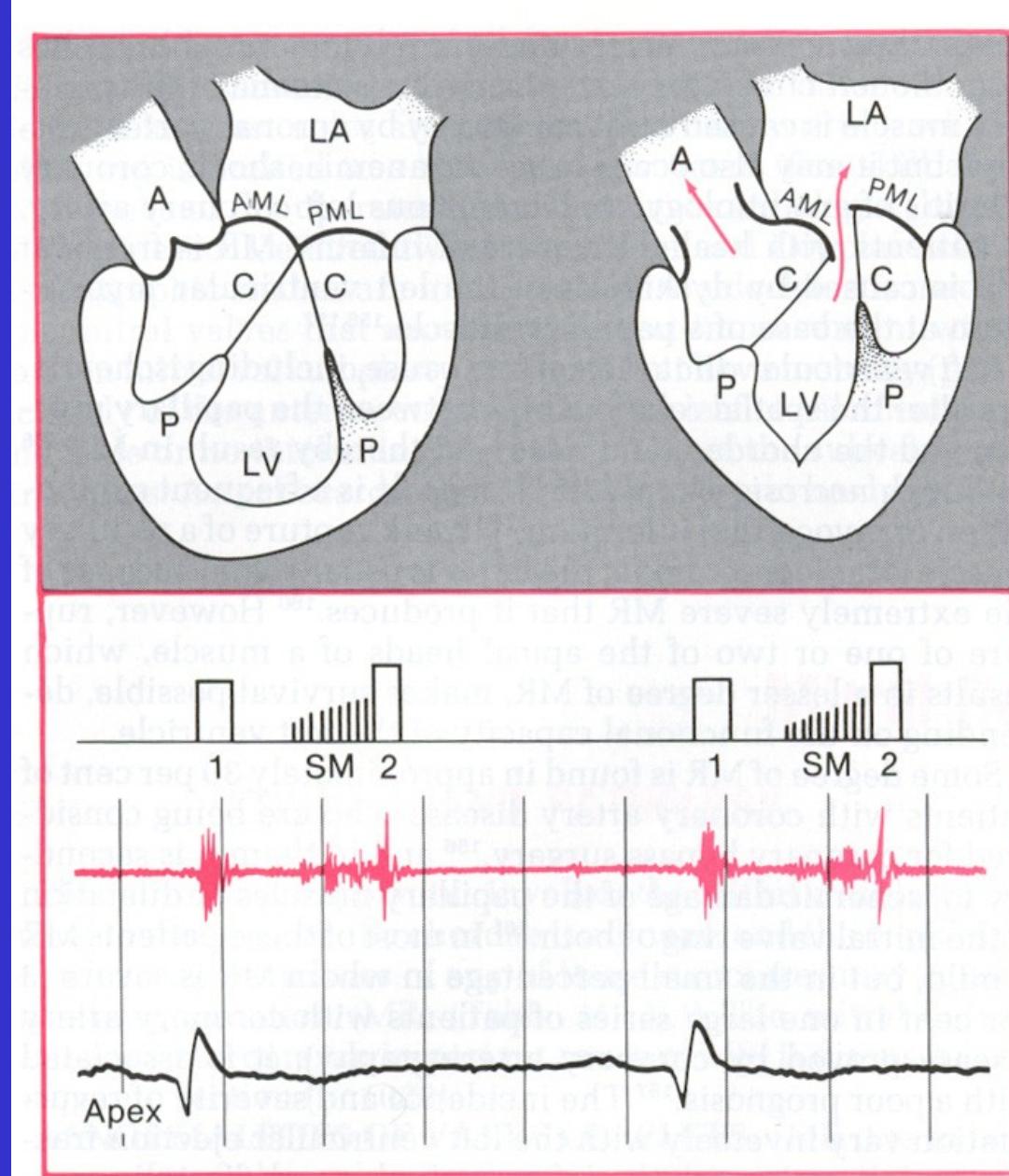
# Pansystolic Murmur, LV and LA pressures during Chronic Mitral Regurgitation



**Mitral Valve Prolapse causes regurgitation due to mismatch of MV leaflets. Note crescendo murmur.**

**Most common form of valvular HDz- 2-6% of population.**

**Life expectancy is normal except in significant MR, severely thickened leaflets, enlarged LV/LA especially men >45**



# MR Treatment

- No medical therapy
- Most difficult clinically
  - By the time symptoms occur, it may be too late
- Drop in EF or development of atrial fibrillation enough to justify surgery

# “Pearls”

- Diastolic murmurs usually represent pathological conditions as do most continuous murmurs.
- Most important issue in patient with a cardiac murmur is the presence or absence of symptoms.
  - Many asymptomatic children and young adults with grade 2/6 midsystolic murmurs and no other cardiac physical findings need no further cardiac evaluation
  - **Many asymptomatic** elderly patients have midsystolic murmurs related to sclerotic aortic valve leaflets, flow into tortuous, noncompliant great vessels
  - Such murmurs must be distinguished from murmurs caused by mild to severe valvular aortic stenosis (AS) which is prevalent in this age group.

# Conclusions

- Valvular heart disease associated with spectrum of presentations
- Recognition prior to the onset of symptoms may be life saving
- Careful physical exam almost always diagnostic

# Recommendations for Echocardiography in Asymptomatic Patients With Cardiac Murmurs

- 1. Diastolic or continuous murmurs. Class I
- 2. Holosystolic or late systolic murmurs. I
- 3. Grade 3 or greater midsystolic murmurs. I
- 4. Murmurs associated with abnormal physical findings
- on cardiac palpation or auscultation. IIa
- 5. Murmurs associated with an abnormal ECG or chest
- x-ray. IIa

*Circulation.*  
1998;98:1949-1984.

# Class III Indications for Echo

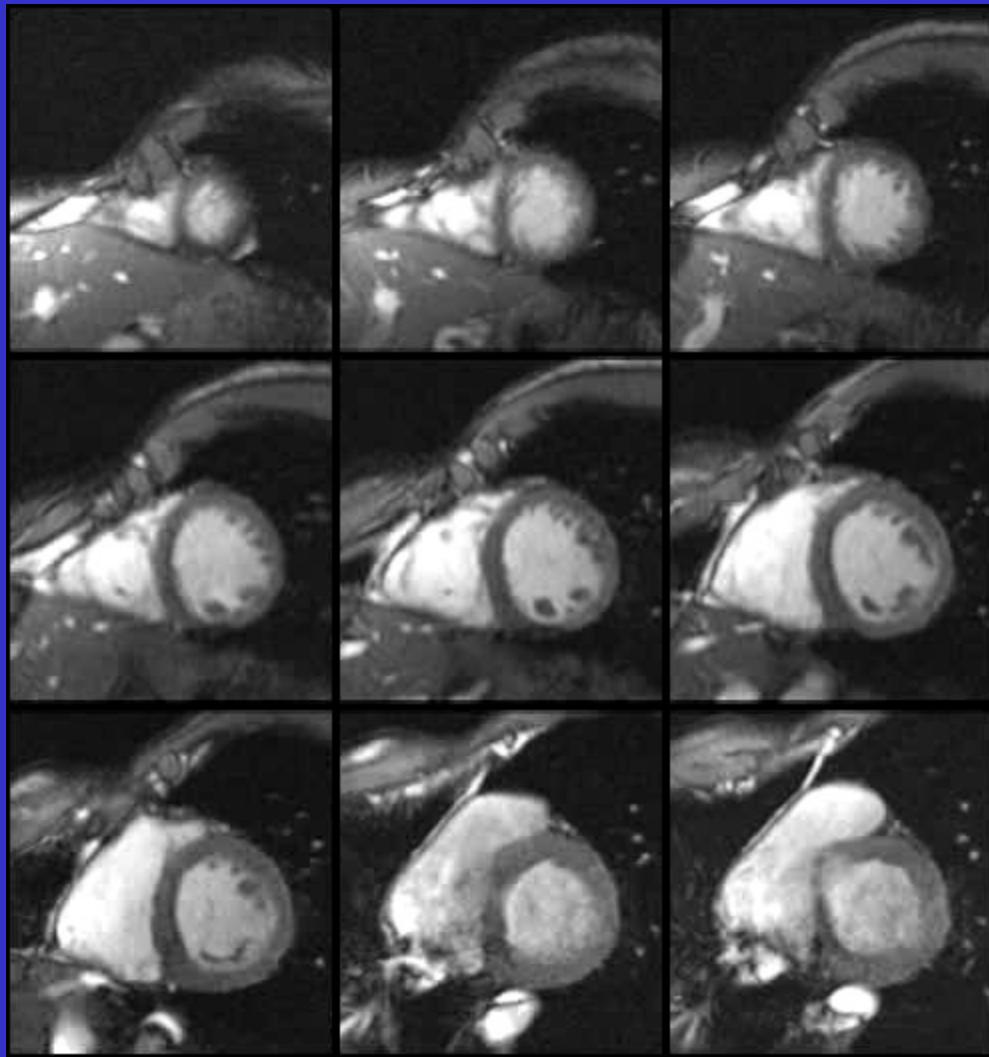
- **6. Grade 2 or softer midsystolic murmur identified as innocent or functional by an experienced observer. III**
- **7. To detect “silent” aortic regurgitation or mitral regurgitation in patients without cardiac murmurs, then recommend endocarditis prophylaxis. III**  
*Circulation. 1998;98:1949-1*

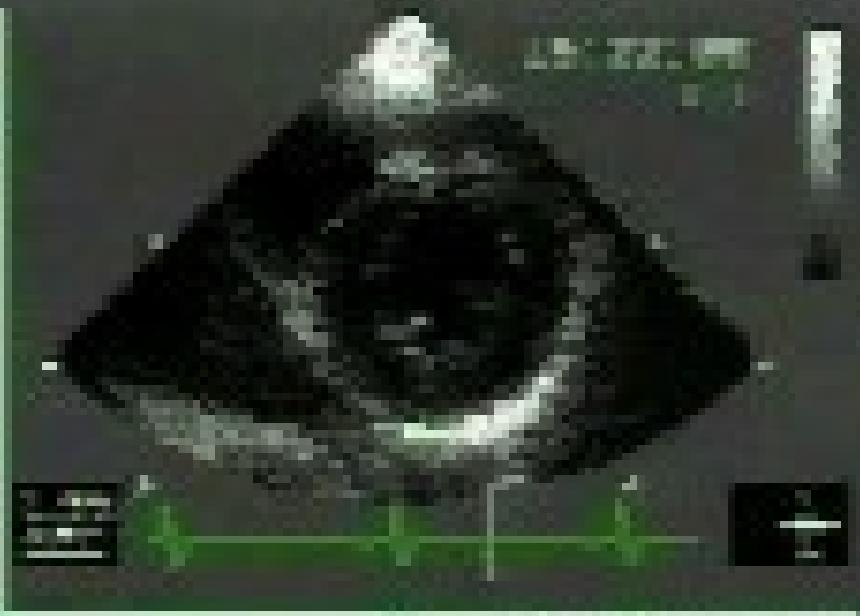
# **Recommendations for Echocardiography in Symptomatic Patients With Cardiac Murmurs**

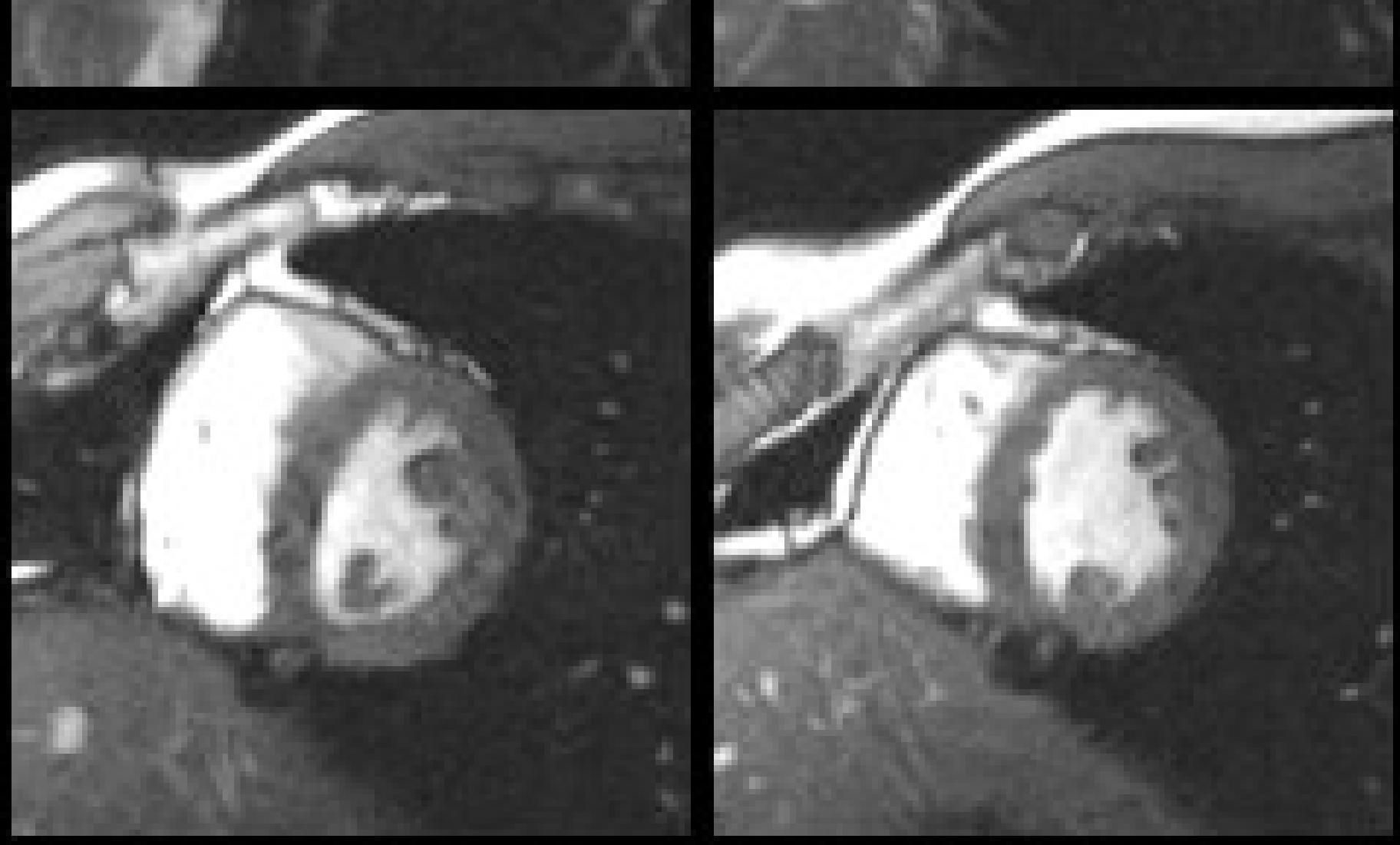
- 1. **Symptoms or signs of congestive heart failure, myocardial ischemia, or syncope.** I
- 2. **Symptoms or signs consistent with infective endocarditis or thromboembolism.** I
- 3. **Symptoms or signs likely due to noncardiac disease with cardiac disease not excluded by standard cardiovascular evaluation.** IIa
- 4. **Symptoms or signs of noncardiac disease with an isolated midsystolic “innocent” murmur.** III





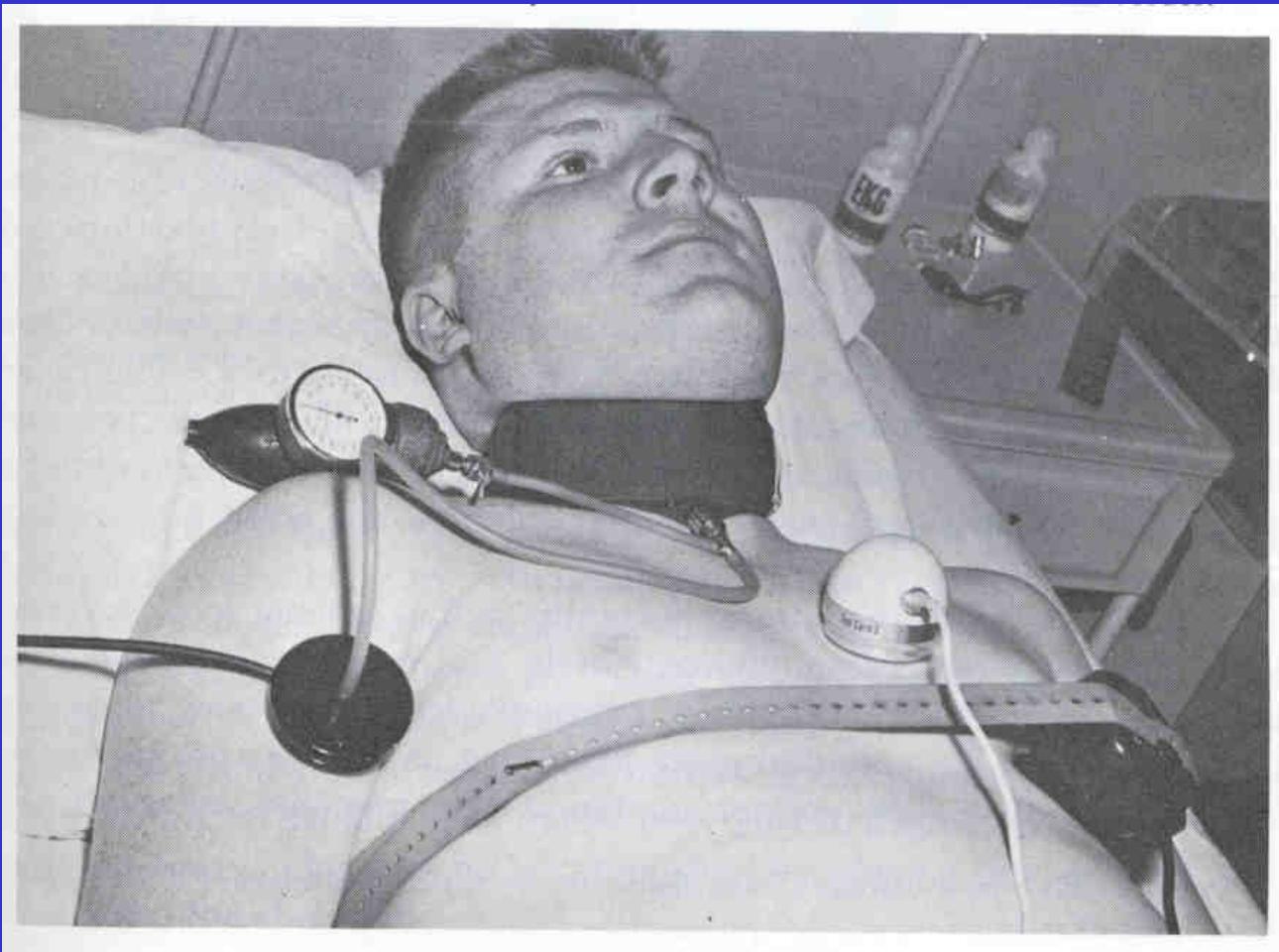






Glockner, et al. *Radiographics*.  
2003;23:e9-e9.

# Valvular Heart Disease



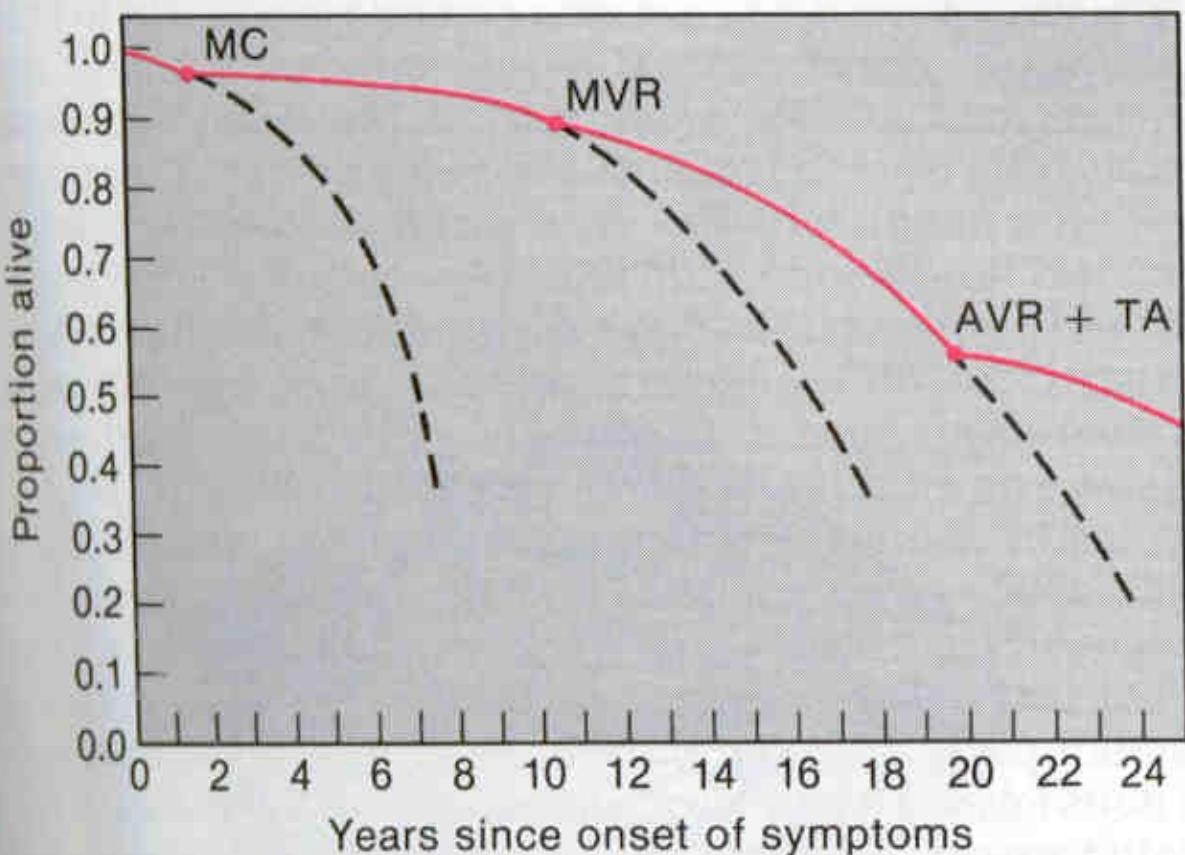


FIGURE 34-6. Schematic representation of the subsequent life history after the initial development of symptoms in a large group of patients with mitral stenosis. The red solid circles and red lines indicate a surgical procedure. The dashed lines represent estimated survival of patients not receiving the surgical procedure. MC = mitral commissurotomy, MVR = mitral valve replacement, TA = tricuspid annuloplasty, AVR = aortic valve replacement. (From Kirklin, J. W., and Barratt-Boyes, B. G.: Cardiac Surgery. New York, John Wiley and Sons, 1986, p. 328.)